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THE RELATION OF HEART RATE TO INTRACRANIAL PRESSURE

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A rise in intracranial pressure above the level of the systemic blood pressure is followed by a rise of blood pressure and a slowing of the heart [Cushing, 1902]. The rise in blood pressure is independent of the vagus [Cushing, 1902], and of the carotid sinus [Guernsey, Weisman & Scott, 1933]. The cardiac slowing is not due to carotid sinus impulses [Heymans, 1928], but is abolished by section of the vagus [Cushing, 1902].

Although this evidence supports the view that the cardiac slowing is due to a direct stimulation of the vagus centre, the possibility that the slowing is secondary to the rise of blood pressure, acting via the cardio-aortic pressor receptors, has not been eliminated. This paper describes experiments to test this point.

Methods

Cats anaesthetized with ether followed by chloralose (0·056 g. per kg. body weight) were used for all experiments. Blood pressure was recorded by a mercury manometer connected to a cannula in the right common carotid artery. A Hürthle manometer connected to a cannula in the right femoral artery recorded the heart rate. The skull was trephined over the left parietal region, the opening being $\frac{3}{4}$ in. in diameter. The dura was incised and a close fitting rubber bung, with a glass tube passing through, was inserted into the trephine opening. The glass tube was connected to a flask containing Ringer's solution at 38° C. The pump and recorder of a syphygmomanometer were attached to the flask. By pumping in the Ringer's solution the intracranial pressure could be raised over 200 mm. Hg and maintained at this level for 20 min.

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A mercury valve was attached to the carotid cannula by a side tube, so that a rise of blood pressure could be prevented, and the pressure maintained at a constant level. The side tube was clipped off when the valve was not required.

Intracranial pressure was gradually raised to a level averaging 50 mm. Hg above the systemic blood pressure, and heart rate and blood pressure recorded. Approximately 30 sec. was taken in raising the intracranial pressure, which was maintained for 5–20 min. These experiments were then repeated with the mercury valve open, so that no rise of blood pressure occurred when the intracranial pressure was raised. In one experiment, the pressure in the cisterna magna was recorded, and it was found that the pressure in the cisterna did not rise to the level recorded on the syphygmomanometer dial, being approximately 10 mm. Hg lower.

Results

Ten experiments were carried out, and in each case a rise in the intracranial pressure was followed by a rapid rise of blood pressure and cardiac slowing. When the blood pressure was maintained at a constant level, the decrease in cardiac rate was nearly as great as when the blood pressure was allowed to rise.

The average rise of blood pressure, when the mercury valve was not in use, was from 140 mm. Hg to 195 mm. Hg, and the pulse rate fell from 190 to 107 per minute. The average intracranial pressure as recorded by the syphygmomanometer dial was 200 mm. Hg.

Using the mercury valve, the blood pressure did not rise more than 5 mm. Hg from 132 to 137 mm. Hg, but the heart slowed from 182 to 113 per minute.

Fig. 1 shows the results obtained in a typical experiment. The cardiac slowing comes on when the intracranial pressure exceeds the systemic pressure, but is maintained even when the blood pressure rises above the intracranial pressure.

Discussion

These experiments show that the cardiac slowing associated with raised intracranial pressure is independent of the rise of blood pressure. The degree of slowing when the pressure is kept constant is less than obtained in the controls, because there is a loss of blood via the mercury valve when the intracranial pressure is raised. This haemorrhage amounts to some 40 c.c. of blood, and such a loss would cause a cardiac acceleration.

The onset of the cardiac slowing was similar in both sets of experiments, coming on when the intracranial pressure exceeded the systemic blood

pressure level. At this point there must be an anaemia of the medulla, but since the cardiac slowing persists after the blood pressure has risen above the intracranial pressure, it is unlikely that anaemia is the cause.

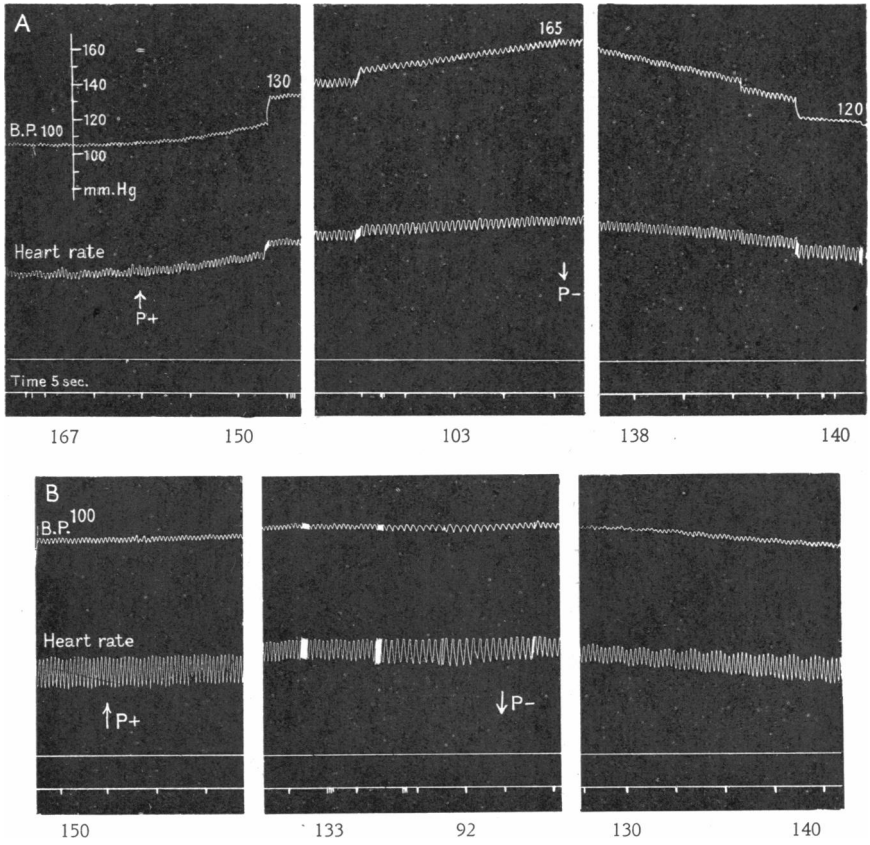

Fig. 1. Record of blood pressure and heart rate. At points marked $P+$ intracranial pressure raised to 170 mm. Hg, at $P-$ the pressure is lowered. The intracranial pressure changed over a period of 35 sec. To obtain the figure for the pressure exerted on the medulla, 10 mm. Hg should be deducted (see text). In the lower tracing, the mercury valve is in use, keeping the blood pressure constant. The heart rate per minute is given underneath the tracings.

Although the intracranial pressure, as recorded by the syphygmomanometer, was in many cases higher than the systemic pressure reached, the experiment in which the pressure in the cisterna magna was measured showed that the pressure here was 10 mm. Hg lower, and if this correction is used, in all cases the systemic blood pressure is found to rise above the

intracranial pressure. This was found by both Cushing (1902) and Eyster, Burrows & Essick (1909).

Pressor receptors cannot be responsible for the cardiac slowing, and it is concluded that the raised intracranial pressure stimulates the vagus centre directly.

SUMMARY

1. The cardiac slowing associated with raised intracranial pressure has been investigated in cats.

2. Preventing the rise of blood pressure produced by a raised intracranial pressure does not abolish the cardiac slowing.

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